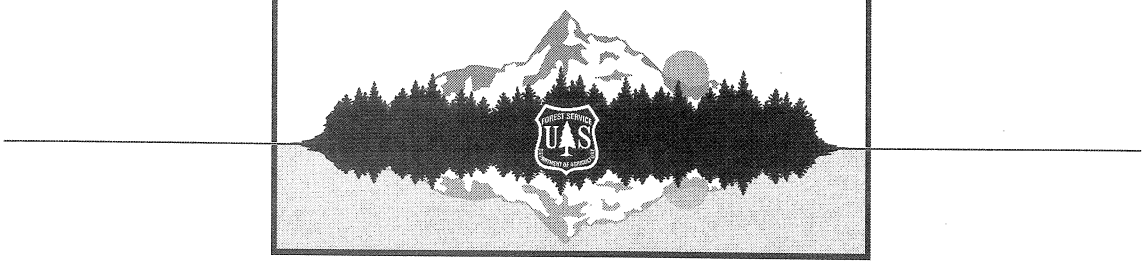
ATTACHMENT 2: ROTENONE TOXICITY TO WILDLIFE

DIAMOND LAKE RESTORATION



FINAL PROGRAMMATIC ENVIRONMENTAL IMPACT REPORT (SUBSEQUENT)

ROTENONE USE FOR FISHERIES MANAGEMENT -JULY 1994-

STATE OF CALIFORNIA
THE RESOURCES AGENCY
DEPARTMENT OF FISH AND GAME

al. 1982). It dissipates quickly under by volatilization, less so by oxidation and very slowly by hydrolysis. These properties parallel CDFG monitoring results; TCE has not been detected in flowing water treatments of 1 mg/L formulated rotenone, but has persisted at or above detectable levels for up to approximately three weeks in impoundments. (Finlayson and Harrington 1991; CDFG 1991a; 1992a; 1992b.)

The half-life of xylene ranges from 3.2 hours to several days (Lyman et al. 1982; Anonymous 1988; MacKay and Leinonen 1975) Xylene levels in water dissipate primarily by volatilization (Anonymous 1988). CDFG monitoring results reflect this; xylene has not been detected in flowing water treatments, but has persisted at or above detectable levels for up to two weeks in impoundments (Finlayson and Harrington 1991; CDFG 1991a; 1992b).

No studies were found on the fate of 2-methylnaphthalene in aquatic systems. However, CDFG monitoring results show that it persists for no longer than 24 hours in flowing waters and up to two weeks in impoundments (Finlayson and Harrington 1991; CDFG 1991a; 1992a; 1992b).

Environmental fate data and CDFG experiences indicate that xylene, 2-methylnaphthalene, TCE, and naphthalene will dissipate before rotenone in impoundments. Naphthalene and 2-methyl naphthalene may persist below a project site in a flowing water treatment for a short duration. Therefore, environmental exposure to these constituents is short-term.

3.1.10 Environmental Fate of Potassium Permanganate

The oxidation rate of rotenone by potassium permanganate is significantly influenced by water temperature. CDFG laboratory studies (1993a) and field experiences suggest that permanganate levels of 2 to 4 mg/L oxidize rotenone from 1 mg/L Nusyn-Noxfish^{R} within 15 and 30 minutes at water temperatures of 15°C and 5°C water, respectively. During rotenone oxidation, potassium permanganate is reduced to manganese oxide, a biologically inactive compound (Engstrom-Heg 1971). In flowing water treatments, this usually limits aquatic exposure to permanganate and rotenone to 0.25 to 0.5 miles downstream of the introduction of permanganate (detoxification site). On one occasion, permanganate caused fish mortalities downstream of a treatment site. In 1992, fish were inadvertently killed downstream of the detoxification site in Silver King Creek (CDFG 1992a).

3.2 Toxicology of Formulated Rotenone

3.2.1 Rotenone Mode of Action for Mammalian, Aquatic, and Avian Species

Rotenone acts as a powerful inhibitor of cellular respiration in fish, mammals, and birds. The site of action is located in the flavoprotein region of the respiratory enzyme chain;

rotenone blocks a link situated at the diaphorase level in the electron transport system (Lindahl and Oberg 1961). Oberg (1964) found that glutamate respiration of brain mitochondria from rotenone-treated fish was significantly lower than untreated fish. The specialized structure of the gills in fish and some aquatic invertebrates favors entrance of rotenone (due to high lipid solubility) into the blood stream; the toxicant is then transported to vital organs and inhibits cellular respiration (Oberg 1967a). Rotenone binds at two specific sites on the reduced nicotinamide adeninedinucleotide (NADH) molecule (Teeter et al. 1969). In bluegill *Lepomis macrochirus* liver mitochondria, rotenone inhibited oxygen uptake. Rotenone-affected bluegill showed glutamate respiration lowered in liver and brain mitochondria by 55 to 80%, respectively (Hiltibran and Johnson 1965). In the gills of treated fish, there is a reduction of gill cell respiration and disruption of chloride cell mitochondria and thus, the ion transporting ability of these cells is impaired (Oberg 1967b).

In part, the selective toxicity of rotenone to fish over mammals is due to the site of action and ease of degradation and not from differences in the two NADH systems (Yamamoto 1970). Mammals are not highly susceptible to orally administered rotenone because they are protected by effective oxidizing enzyme systems.

3.2.2 Fish and Amphibians

The 24-h acute toxicity of rotenone varies from 27 µg/L Noxfish^R for lake trout *Salvelinus namaycush* to 665 µg/L Noxfish^R for black bullhead *Ictalurus melas* (Table 5). Generally, salmonids are the most sensitive, centrarchids less sensitive, and ictalurids the least sensitive to rotenone. Adult amphibian toxicity values range from 5.8 mg/L Noxfish^R (96-h LC_{50}) to 24.0 mg/L Noxfish^R (24-LC_{50}) for frog *Rana pipiens* (Farringer 1972). Frog tadpoles are more sensitive. The 96-h LC_{50} value for Southern leopard frog larva *Rana sphenocephaly* is 0.50 mg/L Noxfish^R (Chandler and Marking 1982).

3.2.3 Aquatic invertebrates

Aquatic invertebrate toxicity values for Noxfish^R (24-h LC_{50}) range from 28.0 µg/L for the cladoceran *Daphnia pulex* to 47.2 mg/L for crayfish *Orconectes immunis* (see Table 6).

3.2.4 Mammals and Avians

Acute toxicity to birds and mammals

Avian acute oral toxicity LD_{50} values for rotenone range from 113 mg/kg for nestling chipping sparrows to >2000 mg/kg for 3-month old mallard ducks (see Table 7). In general, young birds are about 10 times more sensitive to rotenone poisoning than older birds.

Mammalian acute oral toxicity LD_{50} values for rotenone range from 39.5 mg/kg for female rats to 1,500 mg/kg for rabbit (see Table 7).

Santi and Toth (1965) reported very large differences in LD_{50} values between oral and parenteral routes. A possible explanation for this would be the oxidation of rotenone in guts of mammals and birds. This also explains why fish, that have been exposed directly to rotenone at the gills, are much more sensitive than mammals and birds, that have been exposed by oral intake. For example, the oral LD_{50} to rats is 60 mg/kg, while than when given as intraperitoneal is 2 mg/kg. The oral LD_{50} value to dogs is 3,000 mg/kg, while that when given intravenously is 0.65 mg/kg.

Estimates of rotenone's oral toxicity to humans are 300 to 500 mg/kg (Gleason et al. 1969). Tilemans and Dormal (1952) estimated the oral LD_{50} to humans at 2,850 mg/kg, and Hayes (1979) considers an intake of 0.7 mg/kg/d as safe for humans for short time periods.

Chronic toxicity to mammals and avians

Rats fed at 600 to 1200 mg/kg of derris powder (0.06 to 9.6% rotenone) for 200 days exhibited growth depression (Ambrose et al. 1942). Hansen et al. (1965) fed rats at 50 to 11,000 ppm cubé powder in food for two years and fed dogs at 50 to 400 ppm cubé powder in food for 28 months. Rats at all dietary levels, except 50 ppm, exhibited decreased growth, but the incidence of mammary tumors, nephritis, and pituitary lesions was less than controls at all treatment levels; dogs exhibited no clinical or hematological effects for all dietary levels of rotenone tested.

Ellis et al. (1980) administered rotenone to dogs for 6 months at 0.4, 2, and 10 mg/kg/d via gelatin capsules. The 2 mg/kg/d and 10 mg/kg/d dose produced effects of the gastrointestinal tract causing diarrhea, decreased food consumption, and weight loss. Mild anemia and a small decrease in blood glucose, total lipids, and cholesterol was seen at 10 mg/kg/d rotenone. The 0.4 mg/kg/d had no observable effects on the dog. None of the treatment levels produced any histopathologic effects in the skin, spleen, lung, epididymis, pituitary, thyroid, or spinal cord.

Brooks and Price (1961) orally administered 25 to 50 ppm rotenone to Peking ducks and white rock chickens for more than 30 days with no observed toxic effects.

Tisdel (1985) conducted a two-year dietary study in Fisher 344 rats. Diets contained 0, 7.5, 37.5, and 75 ppm rotenone. Significantly reduced terminal body weight were observed in the 37.5 and 75 ppm groups. Females in the mid- and high-dosage

Table 5. Toxicity of Noxfish® (in µg/L) to fish in standardized laboratory tests (Marking and Bills 1976).

Species	LC_{50} and 95% Confidence Interval	
	24-h	96-h
Bowfin *Amia calva*	57.5 50.4-65.5	30.0 23.7-38.0
Coho salmon *Oncorhynchus kisutch*	71.6 63.1 - 81.3	62.0 51.8 - 70.2
Chinook salmon *Oncorhynchus tshawytscha*	49.0 44.3-54.2	36.9 33.9-40.2
Rainbow trout *Oncorhynchus mykiss*	68.9 56.2-84.4	46.0 32.6-64.9
Atlantic salmon *Salmo salar*	35.0 29.7-41.2	21.5 15.5-29.8
Brook trout *Salvelinus fontinalis*	47.0 42.2-52.3	44.3 41.1-47.7
Lake trout *Salvelinus namaycush*	26.9 19.8-36.5	26.9 19.8-36.5
Northern pike *Esox lucius*	44.9 31.4 - 64.3	33.0 26.6-41.0
Goldfish *Carassius auratus*	--	497 412-600
Carp *Cyprinus carpio*	84.0 74.7-94.4	50.0 41.1-60.8

Table 5. (Continued-2)

Species	LC_{50} and 95% Confidence interval	
	24-h	96-h
Fathead minnow *Pimephales promelas*	400 291-549	142 115-176
Longnose sucker *Catostomus*	67.2 59.3-76.1	57.0 51.9-62.6
White sucker *Catostomus commersoni*	71.9 64.0-80.8	68.0 54.0-85.6
Black bullhead *Ictalurus melas*	665 516-856	389 298-507
Channel catfish *Ictalurus punctatus*	400 234-684	164 138-196
Green sunfish *Lepomis cyanellus*	218 197-241	141 114-174
Bluegill *Lepomis macrochirus*	149 124-178	141 133-149
Smallmouth bass *Micropterus dolomieui*	93.2 85.1-102	79.0 70.7-88.2
Largemouth bass *Micropterus salmoides*	200 131-305	142 115-176
Yellow perch *Perca flavescens*	92.0 80.1-106	70.0 59.8-82.0

Table 6. Aquatic toxicity of Noxfish[R] (in mg/L) to aquatic invertebrates in static tests (Chandler and Marking 1982; Farringer 1972; Rach et al. 1988).

Organism	LC_{50} (95% confidence interval) 24-h	96-h
Flatworm *Catennula* sp.	5.10 (3.70-7.03)	1.72 (1.15-2.57)
Cladoceran *Daphnia pulex*	0.0275 (0.0239-0.0316)	-- --
Cladoceran *Daphnia magna*	3.7 (48h EC_{50})	--
Ostracod *Cyprinopsis* sp.	0.490 (0.299-0.803)	0.340 (0.280-0.557)
Freshwater prawn *Palaemmonetes kadiakensis*	5.15 (4.44-6.00)	1.12 (0.760-1.65)
Dragonfly naiad *Macromia* sp.	4.70 (1.45-15.2)	1.00 (0.730-1.59)
Backswimmer *Notonecta* sp.	3.42 (2.27-5.15)	1.58 (0.727-3.44)
Caddisfly *Hesperophylax* sp.	15.0 (9.8-19.0)	2.5 (1.2-6.3)
Caddisfly larva *Hydropsyche* sp.	--	0.605 (0.329-1.17)
Whirligig beetle adult *Gyrinus* sp.	3.55 (2.05-6.15)	0.700 (0.400-1.21)
Snail *Physa pomilia*	6.35 (5.61-7.19)	4.00 (3.45-4.63)
Snail *Oxytrema catenaria*	--	1.75 (1.00-3.06)

Table 6. (Continued-2)

Organism	LC_{50} (95% confidence interval) 24-h	96-h
Snail *Helisome* sp.	30.0 (24.1-37.3)	7.95 (4.63-13.7)
Buckley's filter clam *Elliptio complanto*	--	2.95 (2.23-3.90)
Flattened filter clam *Elliptio complanata*	--	2.00 (1.53-2.61)
Asiatic clam *Corbicula manilensis*	--	7.50 (5.74-9.81)
Crayfish *Orconectes immunis*	47.2 (30.2-61.3)	1.2 (0.4-3.8)
Phantom midge larvae *Chaoborus punctipennis*	--	1.1

Table 7. Acute oral toxicity of rotenone to birds and mammals (from Schnick 1974).

Species	LD_{50} (mg/kg)	Reference
Nestling English song sparrow	130	Cutcomp 1943b
" chipping sparrow	113	" "
" English sparrow	200-850	" "
" American robin	200	" "
Young and old pheasant	850-1,200	" "
3-month-old pheasant	>1,414	Tucker and Crabtree 1970
" " " mallard	>2,000	" "
5-day-old chicken	996	Cutcomp 1943b
Rabbit	>940	Negherborn 1959
White Mouse	350	Kenaga and Allison 1971
Rat	170-245	Lightbody and Matthews 1936
Rat	132	Lehman 1951
Rat-Female	39.5	Eisenman and Thakur 1984
Rat-Male	102	Eisenman and Thakur 1984
Albino rat	130	Cohen et al. 1960
Guinea pig	60	Cohen et al. 1960
Dog	>200 <3,000	Cohen et al. 1960

groups also exhibited significantly reduced food consumption and higher serum urea nitrogen levels. Lower total protein and albumen levels were reported for 75 ppm group females also.

Teratogenicity

Raltech Scientific Services (1981) found that rotenone (98.2% technical grade) does not appear to be teratogenic when orally administered to pregnant mice on days 6 through 17 of gestation at doses of 3, 9, and 15 mg/kg/d rotenone in corn oil. There were no significant differences in copora lutea or implants, implantation efficiency, litter size, sex ratio, mean live fetal weights or lengths, or in the number or percent of live, resorbed, or dead fetuses. No dose-related effects were observed at any level tested. When the results of this study and the range-finding study were interpretated together, the NOEL for maternal effects (weight loss and mortality) and fetal effects (decreased litter size) were 15 mg/kg/d. The A:D ratio is equal to one. Therefore, rotenone is not considered to be a developmental toxicant in mice at levels lower than those that cause maternal toxicity.

Hazleton Raltech (1982a) found that rotenone does not appear to be teratogenic or fetotoxic when orally administered to pregnant rats at doses of 0, 0.75, 1.5, 3.0, and 6.0 mg/kg/d rotenone in corn oil on day 6 through 19 of gestation. Decreased maternal body weights and body weight gains were observed at the 6.0 mg/kg/d level. The NOEL for maternal effects was 3.0 mg/kg/d. There were no significant differences in the number of copora lutea or implants, litter size, implantation efficiency, sex ratio, mortality, resorbed or dead fetuses. At 6.0 mg/kg/d, mean fetal weights were reduced and increased incidences of unossified sternabrae, renal cavitation, and distended ureters were observed in the fetuses. The A:D ration was equal to one. Therefore, rotenone is not a developmental toxicant at levels lower than those that cause maternal toxicity. The NOEL was 3.0 mg.kg/d.

Khera et al. (1982) fed rotenone (87% rotenone and 13% other cubé extractives) at 2.5, 5.0 and 10 mg/kg/d in corn oil to Wistar rats at days 6 through 15 of gestation. The 10 mg/kg/d dose killed over 50% of the parent rats. No other toxicity data are given from the female parents. Rotenone was associated with an increased number of non-pregnant rats and resorptions at the 10 mg/kg/d dose. At 5 and 10 mg/kg/d doses, there were increased incidences of extra rib and delayed ossification of sternebrae, but these effects may have been due to rotenone-induced lethal effects in the female parent.

In a study on reproductive responses to rotenone to Sprague-Dawley rats fed 0.7, 7.1, 14.1, 15.9, 26.0, 32.8, and 40.9 mg/kg/d on days 6 through 15 of pregnancy, Spencer and Sing (1982) noted that fetal survival was reduced at rotenone intake levels $\geq$ 7.1 mg/kg/d in diet but this effect was not dose-

independent. However, pregnant rats given rotenone at ≥ 7.1 mg/kg/d had significantly lower body weights than controls. The weights of the fetuses delivered from dams fed rotenone were not affected. The fetotoxic effects seen in this study may have been caused by toxicity to female parent.

Reproduction

Hazleton Raltech (1983) determined the effects of rotenone on reproductive function and development in two successive generations of rats continuously exposed to rotenone (97 to 98% technical) for 32 weeks at 7.5, 37.5 and 75 ppm of their diet. At week 13, there was a dose-related decrease in body weight of parental male and female (F_0) at rotenone doses of 37.5 and 75 ppm. Rotenone did not have any adverse effect on the reproductive function of either sex at any of the doses. At the 75 ppm dose level, mean litter size at birth was significantly smaller than controls for both $F1_a$ and $F2_a$ litters. There were no other significant differences in litter data, and no pups had physical or behavioral abnormalities. The 75 ppm dose produced bile duct hyperplasia in females and dilation of the gastric glands. The no observable effect level of this study was 7.5 ppm (0.375 mg/kg/d).

Mutagenicity

Goethem et al. (1981) conducted an *Escherichia coli* DNA repair test to screen for rotenone's potential genetic toxicity. Both the spot and liquid suspension tests were used; the spot test was an unsuccessful assay due to the size and solubility of rotenone. The suspension test showed that rotenone up to 10 ppm (highest dose tested) caused no DNA modifying activity; higher doses could not be tested because of precipitation of rotenone. Biotech Research (1981) determined, through cytogenetic analysis of bone marrow cells of rats treated with rotenone, that rotenone was not clastogenic and did not cause chromosomal breaks. Litton Bionetics (1982) found that rotenone was not active in either microbial tests for mutation or mitotic recombination and failed to produce evidence of somatic mutation in the mouse spot tests. The mouse spot tests used embryonic melanocyte cells and the eukaryotic microbial tests used diploid and haploid yeast strains.

The National Academy of Sciences (NAS) reported that no scheduled DNA synthesis was observed in human fibroblast cultures in the presence and absence of a rat liver enzyme activation system when rotenone was tested at concentrations of up to 1,000 Nm (NAS 1983). Negative results were obtained in a rat UDS hepatocyte assay (NAS 1983).

Carcinogenicity

Hansen et al. (1965) exposed rats and dogs for 24 months and 28 months, respectively, to up to 1,000 and 400 ppm cubé, respectively, in the diet with no increase over controls in

mammary tumors, nephritis, and pituitary lesions. Likewise, Innes et al. (1969) fed 3 mg/kg/d rotenone for 18 months to mice with no increase in tumor incidence. Leber and Pershing (1978) exposed Syrian golden hamsters to diets of up to 1,000 rotenone (98% pure) ppm for 18 months with no significant increase in incidence of tumors. Leber and Hake (1978) dosed Sprague-Dawley rats as intraperitoneal and orally with rotenone (98% pure) up to 3 mg/kg/d for 6 weeks and found that it caused no tumors in rats; the rats were observed for 12 months following dosing.

Abdo (1983) reported a two-year dietary study, with concentrations 0, 38, and 75 ppm in Fisher 344 rats. A statistically significant increase in the incidence of parathyroid adenomas, 4 out of 44 individuals in the 75 ppm group compared to 1 out of 41 individuals in controls was observed in males. In a two-year study conducted by Tisdel (1985) in Fisher 344 rats, dietary concentrations of 0, 7.5, 37.5, and 75 ppm rotenone produced no parathyroid adenomas at any level tested and significantly reduced the incidences of pituitary adenomas in mononuclear cell leukemias. Additionally, rotenone has been shown to be a powerful inhibitor of cancer cells in cultures (Haley 1978).

Gosalvez and Merchen (1973) gave intraperitoneal injections (0.17 μg/kg) of rotenone in sunflower oil and chloroform to Wistar rats which resulted in a production of mammary adenocarcinoma with accentuated interstitial fibrosis. The morphology of tumors revealed malignant areas and some of the tumors were transplantable. The tumors appeared 6 months after the rats had received total dosages of 9.1 ± 1.6 mg rotenone. Gosalvez (1983) exposed Wistar rats to oral administration of up to 100 ppm rotenone dissolved in chloroform. At 5, 10, and 20 ppm, there was a 40% incidence of mammary tumors, but at 50 and 100 ppm, there was no increase in tumor incidence over controls. The U.S. EPA (1980a, 1981, and 1989) concluded that because of the use of chloroform (a suspected carcinogen) in the studies, incomplete presentation of data, and lack of information regarding treatment of control animals and histopathology of tumors, that they cannot be used to characterize the oncogenic potential of rotenone.

3.3 Toxicology of Other Formulated Product Ingredients

3.3.1 Fish and Amphibians

The acute toxicity (LC_{50} values in mg/L) of trichloroethylene (>20), xylene (>10), naphthalene (>1), 2-methylnaphthalene (>150), dimethyl naphthalene (>2), and ethylbenzene (>35) to fish and amphibians are all relatively low (U.S. EPA 1980b, 1980c; Johnson and Finley 1980; Verschueren 1983; Korn et al. 1979).

3.3.2 Aquatic Invertebrates

As with fish, the toxicities of the other formulation constituents to aquatic invertebrates are low. Acute toxicity values (LC_{50}) for xylene, trichloroethylene, naphthalene, and 2-methylnaphthalene are greater than 1.0 mg/L (EPA 1980b, 1980c; Verschueren 1983).

3.3.3 Mammals and Avians

The acute toxicity (LD_{50}) values for 2-methylnaphthalene to birds and mammals range from 6,000 to 8,000 mg/kg; values for piperonyl butoxide are at least 2,000 mg/kg; values for naphthalene are >1,500 mg/kg; values for TCE are at least 2,400 ppm; values for xylene are >4,300 mg/kg; and values for ethylbenzene are at least 3,500 mg/kg (Cosmopolitan Safety Evaluation, Inc. 1979; McLaughlin Gormley King Co. 1983; Sarles et al. 1949; Sarles and Vandegrift 1952; Smyth et al. 1969; Tucker et al. 1982; U.S. EPA 1980b; Verschueren 1983; Draize et al. 1948).

3.4 Toxicology of Potassium Permanganate

3.4.1 Fish

The maximum duration of potassium permanganate exposure to aquatic organisms downstream of the treatment area that can be expected from stream treatments is 96 hours. The 96-h LC_{50} values (Table 8) for potassium permanganate to fish species range from 0.750 mg/L for channel catfish *Ictalurus punctatus* to 3.60 mg/L for goldfish *Carassius auratus* (Marking and Bills 1975). Lawrence (1956) found that potassium permanganate is slightly more toxic in warmer water (temperature of 68°F) than cooler water (temperature of 58°F).

3.4.2 Aquatic Invertebrates

Potassium permanganate concentrations of 5 mg/L or more will have lethal affects on microinvertebrates (McKee and Wolf 1963).

3.4.3 Mammals and Avians

Potassium permanganate acute toxicity to mammals is very low. The rat LD_{50} value was determined at 1.09 g/kg (Smyth et al. 1969).

Detoxification

Potassium permanganate may be used to oxidize rotenone (See Section 3.5.9). Potassium permanganate may be used at the farthest point downstream in flowing water application sites and in impoundments or their outflows. Activated carbon may be used to deodorize potable water at water treatment facilities (See Section 3.5.9).

The U.S. Public Health Service water quality limit for manganese is 0.05 mg/L. This limit has been established on the basis of aesthetic and economic considerations rather than physiological hazards (McKee and Wolf 1963). Manganese cannot be considered a physiological hazard because the normal dietary intake is far higher than the amount that would be tolerated aesthetically in potable water. Additionally, exposure to permanganate is limited by the reduction of the compound to manganese oxide during rotenone oxidation. Manganese oxide is a biologically inactive compound (Engstrom-Heg 1971; Engstrom-Heg and Loeb 1972).

Activated carbon may be used to deodorize rotenone treated waters in water treatment facilities. Cost considerations and efficacy studies on activated carbon use have been discussed (See Section 3.5.9). This method could be used for potable water supply sources where the piscicide cannot be allowed to dissipate naturally with time. This method would ensure rapid deodorization of potable water supplies following treatment.

Monitoring

Monitoring measures may be used to ensure detoxification of treated and downstream waters (See Section 3.5.10). These measures will ensure that affected potable water supplies will not be used until waters have been found to contain no detectable rotenone residues or residues of the other organic constituents of the rotenone formulation. Chemical detoxification and monitoring measures will ensure rapid degradation of rotenone and reuse of potable water supplies as quickly as is feasible.

Dead fish removal and disposal

Dead fish which become a public nuisance will be removed from treatment sites and disposed of properly when requested to do so by another public agency. The dead fish will be disposed of in compliance with applicable laws and regulations. This will reduce the biological oxygen demand resulting from the decomposition of dead fish in treated waterways.

4.3 Fish and Amphibians

4.3.1 Impact

Rotenone

Rotenone toxicity to fish and amphibians has been discussed (See Section 3.2.1). A treatment of 2 mg/L formulated rotenone (100 µg/L rotenone) is anticipated to remain toxic long enough to kill most, if not all, of the fish species present in target waters. However, tolerant species such as bullhead *Ictalurus melas* and carp *Cyprinus carpio* may survive by burrowing in mud to avoid exposure. This temporary loss of fish species and gill-breathing amphibians is a potentially significant unavoidable impact that will be evaluated in site specific documents.

Fish eggs are resistant to rotenone treatments because of an impervious chorion. Therefore, treatments should follow the hatching period of target species to ensure a successful treatment, or a follow-up treatment may be required the next year to remove young-of-the-year fish.

A treatment of 2 mg/L formulated rotenone treatment should have little effect on non-gill-breathing amphibians but will likely kill gill-breathing organisms. In flowing waters, fish and gill-breathing amphibians downstream of the treatment site will be at risk by rotenone residues traveling downstream, unless mitigated. Impoundments and flowing waters will remain toxic to gill breathing vertebrates for up to three weeks and less than four days, respectively.

Other organic materials

The toxicity of formulation constituents other than rotenone to fish and amphibians has been discussed (See Section 3.3.2). The toxicities of trichloroethylene, xylene, naphthalene and 2-methylnaphthalene to fish and amphibians are all relatively low (U.S. EPA 1980b, 1980c; Johnson and Finley 1980; Verschueren 1983), and there are several orders of magnitude higher than expected exposure levels in treated waters (See Section 4.2.1). This indicates very little, if any, additional effects to fish and gill-breathing amphibians beyond those caused by rotenone.

4.3.2 Mitigation Measures

Detoxification

Potassium permanganate may be used to protect aquatic species downstream of the treatment site (See Section 3.5.9). From CDFG experiences, potassium permanganate at 3 to 4 mg/L generally has been effective in oxidizing up to 45 µg/L rotenone and 22 µg/L rotenolone residues (resulting from a 1 mg/L Noxfish^R

treatment) to below detectable levels (2 μg/L) (Finlayson and Harrington 1991). CDFG (1993a) studies found that potassium permanganate detoxifies rotenone 15 to 30 minutes after introduction. Consequently, impacts to aquatic organisms from rotenone will be generally limited to 1/4 to 1/2 mile downstream of the detoxification site when potassium permanganate is used in flowing waters. Some fish and gill-breathing amphibian mortalities are expected in this detoxification zone.

Potassium permanganate is toxic to fish and gill-breathing amphibians at the initial concentrations used for detoxification of rotenone. The maximum duration of potassium permanganate exposure to fish downstream of the treatment area that can be expected for stream treatments is 96 hours. Most fish potassium permanganate toxicity (96-h LC_{50}) values (See Section 3.4.1) are within potential treatment levels ($\leq$ 3.6 mg/L). However, as permanganate oxidizes rotenone, it is reduced to manganese oxide which is a biologically inactive compound (Engstrom-Heg and Loeb 1972). Consequently, only aquatic organisms in the 30-minute travel time downstream (usually 1/4 to 1/2 mile) of the detoxification station are generally affected by permanganate toxicity. Potassium permanganate may cause fish mortality downstream of the 30-minute location when the water is overdosed with permanganate or the permanganate demand of the water is reduced. Permanganate was responsible for a fish loss in a small area downstream of the treatment area during the 1992 treatment of Silver King Creek (CDFG 1992).

Monitoring

Monitoring measures will be used to ensure adequate detoxification of rotenone from treated and downstream waters (See Section 3.5.10). In flowing waters, fish in cages downstream of the detoxification site will be monitored to judge the success of the potassium permanganate to minimize downstream toxicity to aquatic organisms. CDFG (1993b) is developing a method for measuring residual permanganate in water.

Restocking

Once a treatment project has been completed and monitoring sample results indicate that the treated waters have detoxified, the water body will be restocked in accordance with a CDFG Fisheries Management Plan or mitigation measures outlined in a site specific environmental document. Aggressive restocking of catchable sports fish should take place in waters where angler use is high; restocking waters for long-term recovery should be conducted in a biologically sound manner. A representative sample of resident fish from the water body can be collected and kept off site during the rotenone treatment and later used to reestablish those fish in the water body.

Generally, restocking projects are quite successful with a high degree of angler satisfaction (Villa, person. commun). Decomposing fish in the reservoir will stimulate phytoplankton production, which will stimulate zooplankton production (Burress 1982). Fish feed extensively on zooplankton. Consequently, in the absence of competing fish species and an abundance of zooplankton, survival and growth rates of stocked fish can be expected to be very high.

4.4 Aquatic Invertebrates

4.4.1 Impact

Rotenone

Rotenone toxicity to aquatic invertebrates has been discussed (See Section 3.2.3). In general, aquatic invertebrates are more resistant to rotenone than fish. However, planktonic crustaceans (microcrustaceans) such as *Daphnia* are very sensitive to rotenone concentrations which kill fish (Hamilton 1941; Smith 1940; Smith 1950).

Some field studies indicate little or no changes in invertebrate populations following formulated rotenone treatments. Two year studies by Houf and Hughey (1973) and Houf (1974) found no short-term or long-term effects on population abundance, relative number of dominant species, or species diversity of either zooplankton or benthos in ponds following treatments of 0.5 to 2 mg/L formulated rotenone. A study by Houf and Campbell (1977) found that benthic invertebrate populations were unaffected by formulated rotenone treatments of 0.5 and 2 mg/L to test ponds.

Most field studies and CDFG experiences indicate that rotenone treatments will kill sensitive aquatic microcrustaceans which will soon be replenished by migration into the treatment area and repopulation of the surviving individuals. Zooplanktonic microcrustaceans have built-in survival responses to environmental stress by laying resting eggs which can withstand extreme hazards including freezing and desiccation . Burress (1982) found that benthic communities were seriously reduced (by 66.5% of previously present species) by a 2 mg/L formulated rotenone treatment, but recovered to higher than pre-treatment levels in 69 days. Studies by Kiser et al. (1963) and Donaldson et al. (1962) resulted in similar findings. A field study of invertebrate populations following a rotenone treatment by Neves (1975) showed that as little as one week may be required for recovery. The cladoceran population in Frenchman Reservoir appeared to have made a strong recovery by August 1991 following the June 1991 treatment of 2 mg/L Nusyn-Noxfish^{R} (R. Decoto, unpublished data). In a creel survey of anglers fishing for planted rainbow trout *Oncorhynchus mykiss* after the treatment of

Frenchman Reservoir, he found stomachs of trout full of *Daphnia spp.* and pupae of midges. Cook and Moore (1969) noticed a rapid and tremendous recovery of organisms (Trichoptera, Ephemeroptera, and Diptera) after sharp reductions of their numbers as a result of a 50 µg/L rotenone treatment (from Pro-Noxfish®, a formulation formally registered in California) to major tributaries of the Russian River, California. Maslin (1988) determined that aquatic invertebrates in Big Chico Creek, Butte County, California, returned to pretreatment levels within six months following treatment with formulated rotenone (2 mg/L Noxfish®) in 1986. Binns (1977) found it took almost two years for sensitive invertebrates to recover from an over-application of rotenone (9 mg/L Chem-Fish Regular®).

Based on this information, a 2 mg/L formulated rotenone application rate will kill most sensitive aquatic invertebrates (cladocerans, hydropsychids, and ostracods) but not affect the least sensitive macroinvertebrates. This loss of invertebrate species is a potentially significant environmental impact. The significance of this impact will be evaluated in site specific documents.

Other organic materials

The toxicities of other formulation constituents to aquatic invertebrates have been discussed (See Section 3.3.2). The toxicity of the other formulation constituents to aquatic invertebrates is low compared to environmental concentrations resulting from a rotenone treatment. Concentrations of these chemicals in the environment resulting from a rotenone treatment will be well below levels toxic to aquatic invertebrates (See Section 3.1.6). Therefore, very few, if any, additional effects to aquatic invertebrates will result from other formulation constituents beyond those caused by rotenone.

4.4.2 Mitigation measures

Detoxification

Potassium permanganate may be used to protect aquatic invertebrates from rotenone toxicity downstream of the treatment site (See Section 3.5.9). From CDFG experiences, potassium permanganate at 3 mg/L has been effective in oxidizing up to 45 µg/L rotenone and up to 22 µg/L rotenolone residues (resulting from a 1 mg/L Noxfish® treatment) to below detectable levels (2 µg/L) (Finlayson and Harrington 1991). CDFG (1993a) found that potassium permanganate detoxifies rotenone (from 1 mg/L Nusyn-Noxfish®) within 15 to 30 minutes after introduction. Consequently, impacts to aquatic invertebrates from rotenone will usually be limited to 1/4 to 1/2 miles downstream of the detoxification site. Some invertebrate mortalities will undoubtedly occur within this detoxification zone.

Potassium permanganate is also toxic to aquatic invertebrates at the concentrations used for detoxification of rotenone (See Section 3.4.2). However, permanganate is reduced to nontoxic manganese oxide as rotenone is oxidized (See Section 3.1.10). Organic matter content and water hardness also limit permanganate concentrations and toxicity. Consequently, only aquatic organisms within the detoxification zone should be affected by potassium permanganate toxicity.

Monitoring

Monitoring measures will be used to ensure detoxification of rotenone from treated and downstream waters (See Section 3.5.10). In flowing waters, the use of monitored fish in cages can be used to judge the effectiveness of potassium permanganate in oxidizing rotenone and thus, minimizing downstream toxicity to aquatic organisms. CDFG (1993b) is also developing a method for measuring permanganate residuals in water.

Recolonization

Recolonization of invertebrates normally begins immediately after detoxification. However, from two to six months may be required for the sensitive, invertebrate populations to reestablish to original abundance and species diversity.

4.5 Avian and Mammalian

4.5.1 Impact

Acute and chronic toxicity of rotenone

The acute and chronic toxicity of rotenone to birds and mammals is low and the impacts are less than significant (See Section 3.2.4). The lowest no-effect levels for rotenone to mammals are 0.4 mg/kg/d and 0.375 mg/kg/d for a chronic dog study and a two generation rat study, respectively. The no-effect level for rotenone to avians is 50 ppm.

The prevailing scientific opinion is that rotenone is not a carcinogen, teratogen, fetotoxin, or mutagen (Leber and Hake 1978; Leber and Pershing 1973; Garrett et al. 1987; U.S. EPA 1980a, 1981, and 1989).

Exposure

At a 2 mg/L formulated rotenone treatment, the maximum rotenone levels present in treated waters would be 100 µg/L and the chemical would be present in diminishing levels for up to 3 weeks.

Mammals and avians may also be exposed to rotenone by consuming rotenone tainted fish following a treatment. Rotenone has been detected in muscle fillets of fish following exposure to Noxfish®. At 2 mg/L Noxfish® (100 µg/L rotenone), 0.045 to 0.101 µg/g rotenone was found in dead channel catfish, largemouth bass, bluegill, and redear sunfish. Black bullhead *I. melas* which survived rotenone at 1.0 mg/L Noxfish® for 11 hours contained 0.05 µg/g rotenone immediately following treatment and <0.020 µg/g rotenone after 12 hours in rotenone-free water (U.S. Fish and Wildlife Service, written communication 1983). Based on these fish residue data, the maximum fish residue that could be expected from a rotenone treatment at 2.0 mg/L formulation would be 0.101 µg/g (ppm). This potential exposure would be further limited by dead fish decomposing or otherwise becoming unavailable for consumption within one week.

Hazard assessment

Using the most sensitive LD_{50} avian value (See Section 3.2.4) of 113 mg/kg rotenone, a 100-g bird would have to consume approximately 100 liters of water (at 100 µg/L rotenone) or more than 100 kg of fish (at 0.101 µg/g rotenone) within a short period of time (24 hours) to receive a lethal dose. A 100-g bird would only be expected to consume 3 to 6 ml of water and about 5 to 10% of its body weight in food (5 to 10 g) daily. Thus, no acute toxicity to birds from drinking water or consuming fish tainted with rotenone (resulting from a treatment of 2 mg/L formulated rotenone) is expected; environmental levels of rotenone are at least 1,000 - 10,000-fold less than that required for lethality.

To exceed the chronic no-effect level for rotenone, a bird would have to have to consume water containing 50 ppm rotenone for 30 days or more. Because environmental levels of rotenone are at least 500 fold less than 50 ppm, the no-effect will not be exceeded. Thus, it is concluded that there will be very few, if any, adverse toxicological effects on avian wildlife resulting from a treatment of 2 mg/L formulated rotenone.

Toxicological hazards to mammalian wildlife from rotenone exposure are also small. The most sensitive oral LD_{50} value was 60 mg/kg rotenone for guinea pigs. This equates to a 100-g guinea pig having to consume 60 liters of treated water within 24 hours to receive a lethal dose. The acute oral LD_{50} value was 3,000 mg/kg rotenone for dogs. This means that a 10-kg dog (or other canine) would have to consume 30,000 liters of treated water at one time to receive a lethal dose. Likewise, a guinea pig and a dog would have to consume in excess of 10 and 300,000 kg of rotenone tainted fish, respectively, to receive a lethal

dose. Thus, no acute toxicity to mammalian wildlife from drinking water or consuming fish tainted with rotenone (from a treatment of 2 mg/L formulated rotenone) is expected.

No chronic toxicity to mammalian wildlife is expected from the use of 2 mg/L formulated rotenone. A 10-kg dog would have to regularly consume 40 liters of water or over 40 kg of fish per day (containing the highest expected rotenone residues) for the 0.4 mg/kg/d chronic no-effect level to be exceeded. A 10-kg dog would be expected to consume less than 2 liters of water or 1 kg of fish per day. Thus, no chronic toxicity to mammalian wildlife from drinking water or consuming fish tainted with rotenone (resulting from a treatment of 2 mg/L formulated rotenone) is expected because exposure levels are at least 20- to 40-fold less than those required to exceed the chronic no-effect level. Furthermore, it is unlikely that a six-month exposure to wildlife would occur because rotenone degrades within 3 weeks or less in water, and fish decompose within one week.

Berteau (1984) suggested that a safety factor of 100 should be applied (10 for variation within species and 10 because the study was done for less than a lifetime) to the no-effect level of 0.4 mg/kg/d to derive an acceptable life-time exposure level of 0.004 mg/kg/d rotenone for dogs. For a 10-kg dog, over 0.4 liters of water or 0.4 kg of fish per day (containing the highest expected rotenone residues) for a life-time would have to be consumed for the 0.004 mg/kg/d rotenone to be exceeded. However, indefinite, life-time exposure could not occur because fish and rotenone residues in water are gone within 1 and 3 weeks, respectively.

It is highly unlikely that wildlife will exceed a short-term lethal dose of rotenone because of the large volume of water or fish which must be consumed. It is equally unlikely that a sub-chronic, no-effect level for rotenone would be exceeded because of the large volume of treated water that must be consumed on an indefinite, basis and because dead fish will rapidly decompose or otherwise become unavailable for consumption. Finally, it is unlikely a life-time "safe" exposure level to rotenone will be exceeded because of the short duration of rotenone in water (maximum of 3 weeks) and availability of rotenone-tainted fish (maximum of 1 week). Thus, it is concluded that there will be no adverse toxicological effects on avian and mammalian wildlife at a treatment of 2 mg/L formulated rotenone.

An indirect effect to fish eating birds and mammals will be a temporary increase in available dead fish immediately following the treatment for several days. During recent CDFG treatments, fish-eating birds (i.e., herons) and mammals (i.e., raccoons) were found foraging on dying and recently dead fish for several days following treatment (Villa pers. comm.).

Following this abundance of dead fish, rotenone treatments will result in a temporary reduction in food supplies for fish- and/or invertebrate-eating birds and mammals. While no detailed information is available on the effects this temporary food reduction has on the food web, there is no indication that significant impacts result to bird or mammal populations. The temporary loss in food resources for animals is a potentially significant unavoidable impact that will be evaluated in site specific documents. It is anticipated, however, that these animals will find other suitable food in the area or move out of the treatment area until fish and invertebrate populations have returned to pretreatment levels.

Other organic materials

The toxicity of formulation constituents has been discussed in Section 3.3.3. The acute toxicity of 2-methylnaphthalene, naphthalene, trichloroethylene, and xylene to birds and mammals are several orders of magnitude higher than the expected levels in the environment resulting from treatment of a 2 mg/L formulated rotenone (See Section 3.1.6). As is the case with rotenone, exposure to these chemicals will not exceed three weeks, and in many cases these materials will dissipate faster than rotenone (See Section 3.1.9). Therefore, chronic exposure to these organic materials to birds and mammals (resulting from a treatment of 2 mg/L formulated rotenone) is not expected.

4.5.2 Mitigation Measures

Restocking

Once a treatment has been completed and the treated waters have been detoxified, the water body will be stocked with fish, usually native California fish species. This will ensure that the loss of fish to a water body will be temporary. This, accompanied with a rapid recovery of aquatic invertebrates, will ensure a replenished food supply for wildlife within one to two months.

There have been several instances were special arrangements were made to accommodate the needs of wildlife impacted by a temporary loss of food resulting from a rotenone treatment. In Hyatt Lake, Oregon, nesting bald eagles and ospreys were protected through a feeding program (Smith 1991). In Frenchman Reservoir, California, the eggs were taken from nesting bald eagles and transplanted to a rehabilitation program on Santa Catalina Island to avoid survival problems (CDFG 1991d).

Project timing

Where possible, rotenone treatments will be conducted in early fall. Since the need for food is greatest in the spring

and early-summer, due to the requirements of care and raising of young, fall treatments should have less impacts on fish and invertebrate dependent animals than a spring or early-summer treatment.

4.6 Threatened and Endangered Species

If a federal or state listed threatened or endangered wildlife species may be affected by a rotenone treatment, mitigation programs (monitoring, alternative food source provision, etc.) will be coordinated with the U.S. Fish and Wildlife Service to ensure no significant impacts to the species (See Section 3.6.3). This coordination was successfully completed when young bald eagles where threatened during the Frenchman Reservoir treatment (CDFG 1991d). Bald eagle eggs were transplanted from Frenchman Reservoir prior to treatment when it was apparent that there would be insufficient aquatic food to feed young-of-the-year birds because of the treatment in the spring. The eggs were successfully transplanted to the Santa Catalina Island Bald Eagle Reestablishment program. In Hyatt Lake, Oregon, nesting bald eagles and ospreys were protected through a feeding program (Smith 1991). If a state listed threatened or endangered species may be affected by a rotenone treatment, mitigation monitoring will be coordinated by CDFG Rotenone Committee (See Section 3.6.2).

4.7 Plants

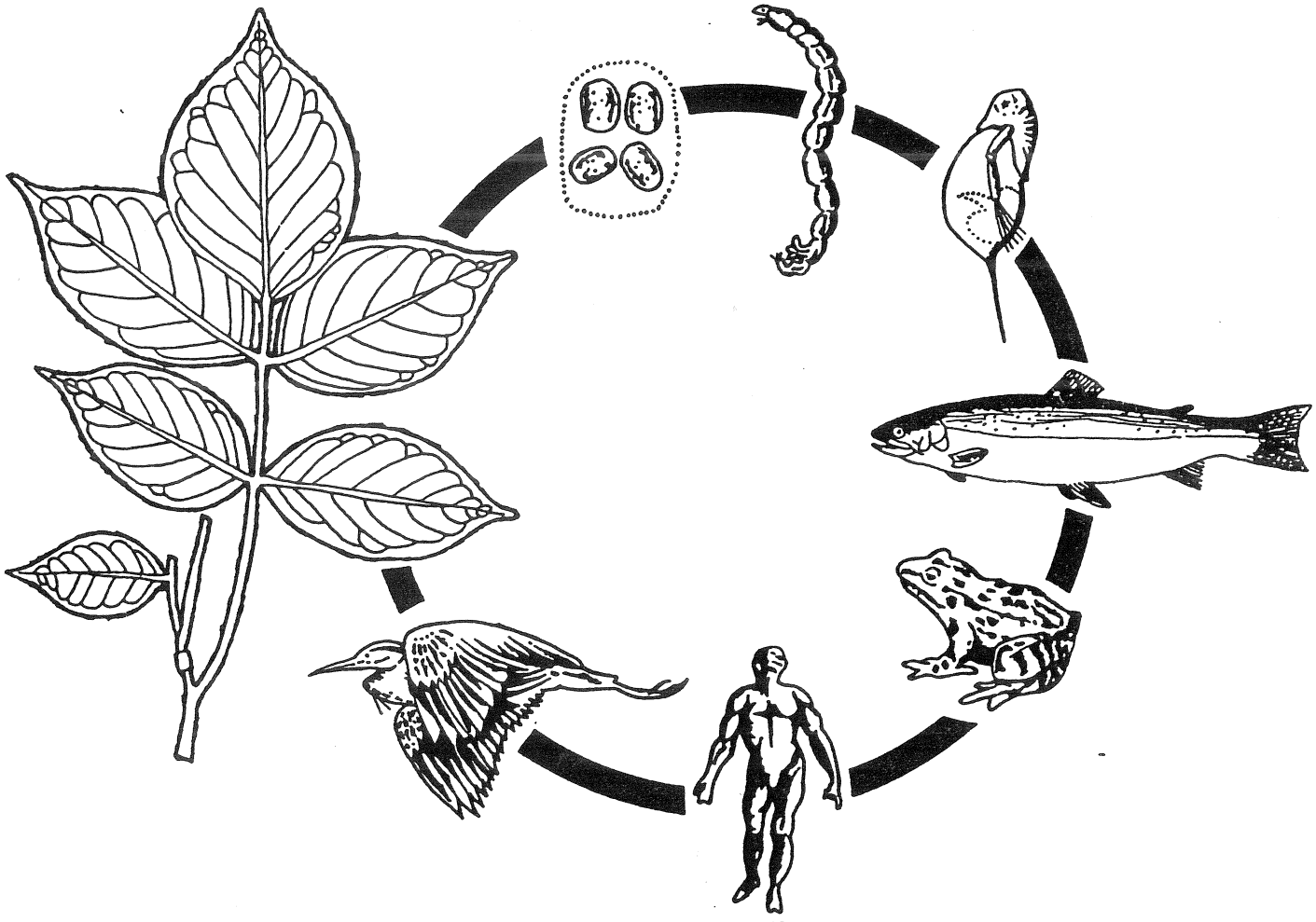
Rotenone formulations are not toxic to plants (U.S. EPA 1989). The only potential effect on plant populations that may result from a rotenone treatment would be direct, physical disturbance, such as trampling by people and equipment or earth movement. This will not cause significant impacts to foliage in the treatment area because few personnel and little equipment are required for most treatments.

Nitrogen fixation in soil, sediment, and water was not greatly inhibited or enhanced by rotenone (Hazelton Raltech 1982b). No substantial effects on nitrification were observed in soil or sediment. However, relatively long-term inhibition of microbial activity in sediment was observed at rotenone residue levels and 5 and 25 ppm. This is not considered important because CDFG monitoring studies (a) usually don't detect rotenone residues (detection limit of 0.050 ppm) in sediment, (b) when detected they never exceed 0.50 ppm, and (c) the residues don't persist for more than ten days (Finlayson and Harrington 1991).

Washington Department of Game

FISHERIES MANAGEMENT REPORT 86-2

ROTENONE AND TROUT STOCKING



A literature review with special reference to Washington Department of Game's Lake Rehabilitation Program

ALEX BRADBURY

ROTENONE AND TROUT STOCKING

A literature review with special reference to Washington Department of Game's Lake Rehabilitation Program

Alex Bradbury

Washington Department of Game
Fisheries Management Division

1986

presence of large grazers.

12) When fish are restocked at former levels, grazing and therefore algal abundance can be expected to return to prerotenone levels. Since there is a net decrease in fish following removal of nontrout species, there may be a decrease in algae.

3.2.5 Effects on Other Animals

Wild and domestic animals that live near treated lakes may be affected by rotenone in two ways:

1) they may ingest rotenone either by drinking treated water or eating dead fish; and
2) the food supply of some may be affected by the temporary loss of fish and aquatic insects following rotenone.

Laboratory toxicity data for mammals, birds, and amphibians are readily available (see below), but no work has been done examining rotenone's indirect effects on riparian animals as in (2) above. To deal with this question we must rely solely on life history data for the animals involved.

Effects on Mammals

Data on the acute toxicity of orally administered rotenone to mammals are listed in Table 25. Only oral LD50's using aqueous solutions are shown, since these mirror the "real-life" situation; see Schnick (1974) for the results of studies involving IP, IV, and IM injections of rotenone, as well as oral doses using unusual solvents.

The lowest LD50 of pure rotenone found in the literature on mammals is 55 mg/kg body weight for guinea pigs (Cutkomp 1943b). To kill a small mammal weighing approximately half a pound would therefore require 12.5 mg pure rotenone, or 250 mg of the commonly-used 5% dust. Since the animal could only come in contact with the rotenone by swallowing lake water, it would have to drink 125 liters (33 gallons) of lake water treated with a 2 ppm dosage. Furthermore, this makes the unlikely assumption that the rotenone has lost none of its potency while in the lake (see Section 3.1.1).

Likewise, the smallest mammalian LD50 of rotenone formulation found in the literature is 170 mg/kg body weight of cubé powder (4.7% rotenone) reported by Haag and Taliaferro (1940) using male rats. A half pound animal would have to swallow 39 mg cubé, or 19.5 liters (5 gallons) of lake water treated with 2 ppm to ingest a lethal dose of rotenone. More recent laboratory work with formulations like Pro-Noxfish put the median lethal dosage of lake water more on the order of 49 gallons for a half-pound mammal (Brooks 1961).

To produce subacute effects such as weight loss or liver damage also requires unrealistically high dosages fed continuously in the diet for many months (Section 3.2.7).

Thus, rotenone would have no direct toxic effect on mammals in either the short or long run. The reasons for the high mammalian tolerance to rotenone were discussed in Section 3.1.1. The U.S. Environmental Protection Agency (1981) considers it safe to water livestock with rotenone-treated water.

Indirect effects might occur when rotenone disrupts the food supply for small mammals that feed on fish or benthos. In Washington, this category includes the mink, river otter, and water shrew.

Mink feed primarily on small mammals, however, with fish a secondary food

Table 25. Median lethal dosages (LD50) of pure rotenone and rotenone formulations administered orally to animals.

Animal	LD50	Formulation	Reference
Rabbits	1.7 ml/kg	Chem-Fish Special Pro-Noxfish	Brooks 1961
White mice	350 mg/kg	pure rotenone	Kenaga and Allison 1971
Rats	1.5±0.1 ml/kg	Pro-Noxfish	Brooks 1961
	170 mg/kg	cubé (4.7% rotenone) in aqueous solution	Haag & Taliaferro 1940
	132 mg/kg	pure crystalline rotenone	Lehman 1951
	1500 mg/kg	derris	Lehman 1951
	1.5 cc/kg	Chem-Fish Special	Blue Spruce Co. 1973
Guinea pigs	60 mg/kg	pure rotenone	Cohen et al. 1960
	55-60 mg/kg	pure rotenone	Cutkomp 1943b

source (Banfield 1974). Moreover, they move frequently, all dens being temporary (Whitaker 1980).

River otters, on the other hand, rely almost entirely on fish for food, and the temporary loss of prey following rotenone poisoning may disturb them. But otters forage widely, sometimes travelling 50-60 miles during a year (Banfield 1974), and would probably not be displaced permanently. Water shrews may be indirectly affected by the temporary reduction in benthos (Doug Wechsler, WDG Nongame Program biologist, personal communication).

Effects on Birds

Oral toxicity for birds is listed in Table 26.

The chipping sparrow is the most susceptible of the birds tested, with an LD50 of 113 mg pure rotenone per kg body weight. A six-ounce chipping sparrow would require 19.2 mg pure rotenone, or 384 mg of the 5% fish-killing dust for a lethal dose; this would require the bird to drink 192 liters (almost 51 gallons) of lake water treated with 2 ppm.

Similar calculations based on Brooks' (1961) work show that the lethal dose for a 6 oz. white rock chicken would be 1.02 ml Noxfish; such an amount would only be found in 510 liters (135 gallons) of lake water treated with 2 ppm Noxfish.

Quite obviously, there would be no direct toxic effect of rotenone on birds. Although no chronic, long-term toxicity studies have been performed on birds, the quick breakdown of rotenone and infrequent poisonings preclude such effects.

As with mammals, only those birds which depend on fish or benthos for food could be affected indirectly by rotenone treatment of a lake. Here, however, the list is longer: bald eagles, ospreys, loons, kingfishers, rails, grebes, and diving ducks - notably mergansers, buffleheads, and goldeneyes - all rely on diets of fish and/or benthos. Except for the kingfisher, all these birds normally forage as adults over many miles and would probably not be harmed by the temporary loss in fish or benthic food following rotenone (Lora Leschner, WDG Nongame Program biologist, personal communication; State of California 1983). Ospreys leave the Pacific Northwest beginning in September, returning in April, and thus would not be present during most fall treatments.

Kingfishers, however, are highly territorial, so that the temporary disappearance of fish could force them off a lake and into competition with birds on other waters (Doug Wechsler, WDG Nongame Program biologist, personal communication). Ducklings on a spring-rotenoned lake would be unable to forage on other waters, and may suffer reduced growth as an indirect result of rotenone treatment. Both kingfishers and ducks live on frequently-rotenoned lakes, so that these losses - if they occur at all - are not absolute. Unfortunately, there are no quantitative studies on this topic.

Effects on Amphibians and Reptiles

Table 27 lists toxicity data for amphibians. No laboratory data are available for reptiles.

Table 26. Median lethal dosages (LD50) of pure rotenone and rotenone formulations administered orally to birds.

Animal	LD50	Formulation	Reference
White rock chickens	6 ml/kg	Chem-Fish Regular Noxfish	Brooks 1961
	8 ml/kg	Chem-Fish Special Pro-Noxfish	
Chickens (4-week)	>270 mg/kg	pure rotenone	Cutkomp 1943b
Chickens (5-day)	996 mg/kg	pure rotenone	Cutkomp 1943a
Chickens (5-day)	247 mg/kg	derris extract (25% rotenone)	
Eastern chipping sparrow (nestling)	113 mg/kg	pure rotenone	
Eastern song sparrows (nestlings)	130 mg/kg	pure rotenone	
Eastern robins (nestlings)	195 mg/kg	pure rotenone	
English sparrows (nestlings)	199 mg/kg	pure rotenone	
English sparrows (adults	853 mg/kg	pure rotenone	
pheasants (5-day)	850 mg/kg	pure rotenone	
pheasants (4-week)	1190 mg/kg	pure rotenone	
pheasants (3-4 month)	>1414 mg/kg	pure rotenone	Tucker & Crabtree 1970
prairie horned larks (adult)	450-500 mg/kg	pure rotenone	Cutkomp 1943a
mallards (3-4 month)	>2000 mg/kg	pure rotenone	Tucker & Crabtree 1970

Table 27. Toxicity of rotenone to amphibians in laboratory bioassays.

Animal	Concentration (ppm)	Exposure	Formulation	Water Chemistry	Comments	Reference
Southern leopard frog larvae (Rana sphenocephala)	0.5	96 hr.	Noxfish	16° C; see Lennon & Walker (1964) for test conditions	LC50	Chandler & Marking 1982
Leopard frog (Rana pipiens)	7.3	24 hr.	Dri-Noxfish	12° C, pH 7.2-7.6, 40-48 mg/l hardness	LC50	Farringer 1972
	7.9	24 hr.	Dri-Noxfish	12° C, pH 7.6-8.0, 160-180 mg/l hardness		
	4.6	96 hr.	Dri-Noxfish	12° C, pH 7.2-7.6, 40-48 mg/l hardness		
	3.2	96 hr.	Dri-Noxfish	12° C, pH 7.6-8.0, 160-180 mg/l hardness		
Leopard frog tadpoles (Rana pipiens)	0.1	8-24 hr.	5% rotenone	---	100% mortality	Hamilton 1941
tiger salamander, with gills (Ambystoma tigrinum)	0.017	8-24 hr.	5% rotenone	---	toxic but not necessarily fatal	
tiger salamander, metamorphosed (Ambystoma tigrinum)	0.1	8-24 hr.	5% rotenone	---	100% mortality	
frogs	4.0 mg/kg body weight	---	pure rotenone	---	oral LD50	Haag 1931

These tests suggest that larval amphibians such as tadpoles are far more susceptible to rotenone than metamorphosed adults. This stands to reason when we consider rotenone's high toxicity to gill-breathing forms (Section 3.1.1).

The young of many amphibian species have completely metamorphosed and lost their gills by fall, when most rotenone treatments occur (State of California 1983). Others, however, metamorphose during the fall, so that at least some individuals could be affected by rotenone treatment. In Washington, this category includes the spotted frog (Rana pretiosa), the red-legged frog (Rana aurora), the Northern leopard frog (Rana pipiens), the long-toed salamander (Ambystoma macrodactylum), and the roughskin newt (Taricha granulosa). Still others overwinter with gills: the Pacific giant salamander (Dicamptodon ensatus), the Cascades frog (Rana cascadae), and the bullfrog (Rana catesbeiana). The tiger salamander (Ambystoma tigrinum) never loses its gills, while the Northwestern salamander (Ambystoma gracile) is variable: some metamorphose in the fall, some overwinter with gills, and some retain gills for their entire life (Doug Wechsler, WDG Nongame Program biologist, personal communication). Larvae and gill-breathing adults of the above species could potentially suffer from routine fall rotenone treatments. Spring treatments could affect all species, since young amphibians are always in the gilled stage during that time of year.

Laboratory tests, however, indicate that gill-breathing amphibians have a relatively high tolerance to rotenone. Chandler and Marking (1982) reported that larval leopard frogs were 3-10 times more tolerant of rotenone than most of the 21 fish species tested by Marking and Bills (1976), and had about the same tolerance as the hardy goldfish (Section 3.2.1, Table 10). They noted that these animals were more sensitive to rotenone in the lab than in the natural environment, and concluded that they would probably be safe during lake treatments.

Denis and Devlin (1968) found that rotenone inhibited cell respiration and development in amphibian eggs. Lamy and Melton (1972) noted that rotenone produced unusual cleavage in leopard frog embryos. The laboratory procedures used in both these studies make extrapolation to the "real-world" lake environment impossible. Again, however, frog and salamander eggs are not present in the fall when most rotenone treatments occur.

Actual field data involving amphibians and reptiles are scarce and qualitative. When Brown and Ball (1943a) applied 0.5 ppm 5% rotenone dust to a Michigan lake in early May, tadpoles were "greatly affected." Three months later, however, tadpoles were "extremely numerous," and the authors attributed it to post-rotenone breeding and the lack of predation by fish. High concentrations (~10 ppm) of Noxfish applied to ponds in Florida made alligators visibly ill, forcing them to leave the water (Doug Fletcher, WDG biologist, personal communication).

In other field applications, Meehean (1942) noted that numerous salamanders (Pseudobranchus striatus) were killed by 0.5 ppm derris in five Florida lakes. The same author reported that 1.0 ppm derris killed the soft-shelled turtle (Amyda ferox).

Both adult and larval amphibians, as well as reptiles, may be indirectly affected by rotenone treatment. Most of Washington state's riparian herptiles

include fish and/or aquatic insects in their diets (Hodge 1983; Stebbins 1966), though none depend exclusively on these items. Aquatic insect reduction due to rotenone is rarely more than 71% in the studied waters, and full recovery usually occurs within a month or two (Section 3.2.2). Alternative food sources can probably support these animals during the post-rotenone shortage of fish and benthos (State of California 1983), although only quantitative field tests will resolve this question completely.

Summary

1) Wild and domestic mammals will not be affected by drinking rotenoned lake water or eating rotenone-killed fish.

2) River otters may be disturbed by the temporary post-rotenone loss of fish. Permanent displacement is unlikely because the otter forages over many miles. Water shrews may be affected by the temporary reduction in benthos.

3) Birds will not be affected by drinking rotenoned lake water or eating rotenone-killed fish.

4) Since most adult fish- and benthos-eating birds forage widely or migrate before the fall, the temporary loss of fish and benthos from a lake will probably not have serious effects. Territorial kingfishers, however, may be affected, as may ducklings on spring-rotenoned lakes.

5) Larval amphibians and adults of certain species, since they have gills, may be directly affected by rotenone treatments in either the spring or fall. This is balanced by the fact that larval amphibians are relatively tolerant of rotenone.

6) Adult herptiles may be indirectly affected by the temporary loss of aquatic insects and fish following rotenone, though alternative food sources can probably support them during this period.

7) Field applications of rotenone have killed turtles and made alligators visibly ill. No laboratory tests have been made using reptiles.